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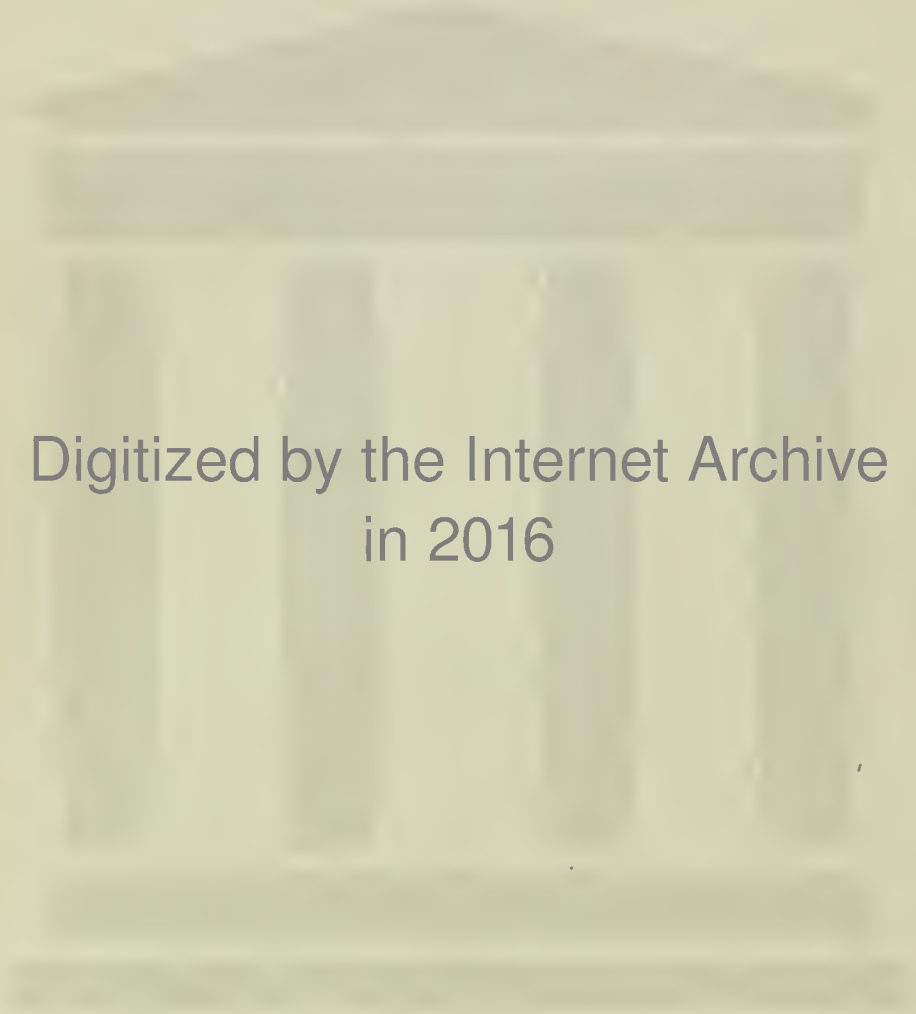

CARDIAC ARRHYTHMIA IN RELATION TO CEREBRAL ANAEMIA AND EPILEPTIFORM CRISES.

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The occurrence of epileptiform seizures in association with cardiac irregularity is by no means infrequent, if we may judge from the literature of the past twenty years, but it is probably comparatively rare in the experience of the individual clinician. And, in view of this fact, it has been thought that the record of a case which presented some remarkable features might be not without interest. The selection of a suitable title has been somewhat difficult, as the majority of distinctions under which the subject has been treated by various observers have given rise to not a little confusion, and, in a few cases, to entire misapprehension. Among the many designations which have found a place in the recent medical journals, we find such as the following:—"Bradycardia and Epilepsy," "Unusually Slow Pulse and Epilepsy," "Epilepsy of Cardio-Vascular Origin." For reasons which will emerge in the course of this paper neither of the two former titles appears to be sufficiently explicit, and probably both are inaccurate. Of the other it may be said that conciseness has been secured at the expense of clearness. The records which form the basis of this paper have been entirely drawn from one case, but that the observations were neither of an isolated nor casual nature may be gathered from the fact that the patient was under observation for a period of nearly two years, and during that

time offered abundant opportunity for the confirmation or correction of previous observations.

The original intention with which the article was undertaken was to endeavour to make a clinical application of Kussmaul and Tenner's experimental work on the causation of epileptiform convulsions, by inducing cerebral anaemia either mechanically or surgically, and that will be the chief object of the paper; but during the progress of the case several other points emerged which call for consideration. There appears to be almost a unanimity among authors who write upon the subject, that when cardiac derangement is associated with convulsions the former stands to the latter as cause to effect; but comparatively few of the contributors to the literature of the subject have accompanied their reports by sphygmographic evidence, which conveys a comprehensive idea of the chief points which they endeavour to demonstrate, and in view of this the accompanying sphygmograms may not be without value. There are two other points upon which anything but unanimity prevails. In the first place the question arises, does true bradycardia or slow pulse exist in association with convulsions? Secondly, when a slow rate of pulse obtains and is associated with convulsions is the action of the heart altogether in abeyance between the powerful contractions, which have a radial expression, or is it making regular although very feeble efforts to contract? Kussmaul and Tenner's work may not possess the attraction which attaches to novelty, but it has the merit of remaining, although not altogether unchallenged, still quite uncontroverted: and in order to a clearer understanding of the subject, some of the more important conclusions arrived at by these observers will be briefly recapitulated.

"The convulsions appearing in profuse haemorrhage (of warm-blooded animals) resemble those observed in epilepsy."

"When the brain is suddenly deprived of its red blood, convulsions ensue, of the same description as those occurring subsequent to the ligature of the great arteries of the neck."

"Epileptic convulsions are likewise brought on when the arterial blood rapidly assumes a venous character, as, for example, when a ligature is applied to the trachea."

"It is highly probable that in these cases the attack of spasms depends upon the suddenly interrupted nutrition of the brain. It is not caused by altered pressure which the brain undergoes."

"The brain of warm-blooded animals can only be deprived of red blood for a short time, otherwise it loses its capability of resuming its functions when again supplied with the nutritive fluid, and the appearance of death becomes a reality. The brain of some rabbits preserved this capability for two minutes."

"Suddenly withheld nutrition is only one of the causes by which the brain is brought into that peculiar internal condition which is manifested in the form of an epileptiform attack."

The writer desires to acknowledge his indebtedness to Professor Samson Gemmell for permission to publish the clinical records of the case.

CASE I. Observed by the Author.

W. T., aet. 48, a weaver by occupation, came under observa- in the Western Infirmary, Glasgow, on September 23rd, 1894, with the complaint of taking what he called weak turns, associated with palpitation, sensations of giddiness, and occasional loss of consciousness, which symptoms had existed for some two weeks previously.

Personal History.—He had never been incapacitated for work by illness at any period of his life prior to the onset of his present illness. There was no history of syphilis, and he had never had rheumatism in any form. In 1892 he suffered from what he called indigestion, and experienced palpitation at night, but this symptom did not obtrude itself upon him during ordinary or unusual exertion. *Occupation.*—In his work as a weaver he was not subjected to any great physical strain, either continuously or otherwise.

History of Present Illness.—About two weeks prior to his admission to the hospital, and while he was standing at his loom and stretching over to tie a thread, he suddenly experienced a sensation of faintness, and a feeling as though he would fall. He remembers nothing of what followed after that for

some seconds apparently. But when consciousness returned he felt that his face was flushed, and his heart was beating forcibly. He was removed to his home, where he remained in bed during the two weeks prior to his coming under observation. With regard to the actual symptoms incidental to this first attack no reliable information can be obtained, but his own feelings and the observations of his friends would seem to indicate that it was almost identical with subsequent seizures. During the interval which elapsed between the occurrence of his first attack and his coming under observation, he has had many similar seizures. Sometimes they seemed to have followed one another in rapid succession, at others whole days have elapsed without the occurrence of a single attack. With regard to the description of these seizures, the information of his wife (who was present at the time of the drawing up of the initial report) is not very full; but she saw him during a seizure after admission to hospital, and she said the attack she then witnessed was identical with those which occurred previously.

When the patient was first seen, the striking fact was the extreme facial pallor, and indeed the patient appeared to be dead, and on palpation of the radial artery no pulse was perceptible. Then one or two beats became perceptible, and gradually increased both in frequency and volume; at the same time the extreme pallor of the face and lips was replaced by very marked flushing. The patient opened his eyes and looked about in a dazed manner as if wondering where he was.

From the sphygmographic tracings which were then taken, it will be seen that there were long intervals during which no pulse was appreciable at the wrist. These intervals lasted sometimes for ten or fifteen seconds. If the finger were kept on the pulse, the following facts were observed and noted in order of their appearance. First, the pulse became suspended: then, and almost at the same moment, the face became rapidly and extremely pale. If the period of asphyxia (here the word asphyxia is used as indicating the absence of the pulse at the radial artery as appreciable to the finger) were only of short duration, say of four or five seconds, there was a gradual and crescendo return of the pulse and a coincident flushing of the face. During these short pauses the patient appeared to be

merely sleeping. Frequently, however, the period of asphyxia lasted much longer, ten, fifteen, and twenty seconds elapsing between the pulse beats. During this time, commencing with the disappearance of the pulse, the pallor of the face became more and more marked; but during the earlier portion of the period of asphyxia nothing special was noted, the patient simply presenting the appearance of death, or breathing in a very shallow manner. Later on, however (perhaps after some fifteen seconds of asphyxia), spasmodic twitchings of the muscles of the face were observed; then, as the period lengthened, the spasmodic movements extended to the muscles of the neck, frequently carrying the head to one or other side, and ultimately the arms and legs took part in the convulsive movements. During the more severe crises, either concomitant squint of both eyes or conjugate deviation to either side was observed. Frequently the eyeballs were turned in an upward and inward direction, and the pupils became widely dilated. When the convulsion was at its height, respiration was noisy, with considerable puffing out of the cheeks and lips and the appearance of froth at the mouth. During the greater part of the period of asphyxia the patient was quite unconscious. The return to consciousness was preceded by the noisy breathing and very marked flushing of the face. On the termination of the convulsion, the patient would look about him in a dazed manner as if just aroused from deep sleep. The flushing of the face and the return to consciousness were always almost simultaneous with the appearance of the pulse at the radial artery, the pulse being felt slightly before the flushing of the face was observed. Auscultation over the precordium, during the asphyxial pauses, discovered what appeared to be abortive beats of the heart, but these beats were not appreciable to the finger at the radial pulse, or indeed in any of the superficial arteries.

Nothing of the nature of Cheyne-Stokes breathing was observed during the crises. During the ordinary or powerful cardiac contractions the second sound, as heard over the pulmonic area, was accentuated.

Examination of the Heart. Area of Precordial Dulness.— The upper border was situated on the fourth left rib. The

left border was just within the nipple line. The right border was on the left margin of the sternum. The apex beat (during the powerful systoles) was visible and tangible in the fourth and fifth inter-spaces, with the point of maximum intensity in the fifth space, about half an inch to the inner side of the vertical line of the nipple.

Lungs.—Percussion and auscultation detected nothing in either lung calling for note.

Liver.—Dulness measured four inches in the vertical nipple line.

Urine.—Was dark amber in colour, with no sediment. The specific gravity was 1015, the reaction was acid, neither albumen nor sugar was detected by the usual tests.

September 25th.—Since admission the paroxysms of asphyxia have been of very constant occurrence, and alternating with periods during which the cardiac action was perfectly regular. A number of tracings taken on the two days following admission showed long periods of asphyxia, the pauses in some cases lasting as long as 15 seconds; one tracing taken during the space of one minute indicated three periods of asphyxia, each of from 12 to 15 seconds' duration, and three periods during which pulsation in the radial artery was recorded—altogether 18 beats being registered in 60 seconds (see tracing No. 2). Another series of tracings taken on the same date showed much shorter periods of asphyxia, 7 or 8 beats being registered in half a minute, and 11 in a whole minute. Most of these beats occurred at regular intervals of 5 or 6 seconds. The terminations of the more lengthy periods of asphyxia were frequently, if not usually, associated with convulsions which occurred when the facial pallor was most profound, and ceased coincidently with the appearance of the facial flushing, and of the radial pulse. During the earlier part of these pauses gradual loss of consciousness was noted, and continued until, and during, the convulsion. The convulsions were, for the most part, more severe than formerly, involving not only the face and neck, but extending to the upper and lower limbs.

Yesterday (*September 24th*) the periods of asphyxia being of frequent occurrence, strychnine in doses of $\frac{1}{50}$ of a grain

was administered hypodermically; brandy in ounce doses given by the mouth; and a synapism applied over the precordium. In the course of half an hour after this treatment was instituted the pulse was found to have become perfectly regular both in its volume and rhythm; the tension was also good.

To-day (*September 25th*).—Regularity of the pulse continued all day, and no tendency to convulsive seizure was observed.

September 26th.—Irregularity of the pulse was again noted this morning, when stimulants and strychnine were administered. As the patient appeared to be in a very critical condition, no sphymographic tracings were taken at this time. In the course of the afternoon there was a marked improvement in the patient's general condition. The pulse became perfectly regular, of good volume, and of excellent tension, as indicated in tracing No. 4.

September 27th.—This morning a new feature presented itself in connection with the character of the pulse. Periods of regularity of volume and of rhythm alternated, with periods when the "pulsus bigeminus" was well demonstrated by means of the sphygmograph (see tracings Nos. 5 and 6), and readily appreciable to the finger. In the afternoon the pulse again became very irregular, the asphyxial pauses were frequent and prolonged, and the patient was practically passing out of one spasm into another. Indeed, the patient appeared to be dying.

September 28th.—The patient's general condition indicates improvement. On auscultation over the precordium, what appears to be a triple rhythm of the cardiac sounds is noted, the first sound apparently being doubled. This fact was observed chiefly over the lower end of the sternum and during the forcible cardiac beats, while during the feeble beats the two reduplicated sounds were audible, but not the single (that is, it was supposed that the doubled first sound was audible, but not the second).

September 29th.—The pulse, as counted at the wrist by the finger, numbered 20 beats to the minute as a rule, but sometimes only 16 beats were registered, and from 16 to 24 were about the limits of variation. Sphygmograms taken to-day also demonstrate characters not previously observed; hitherto no break in the down-stroke or diastolic part of the tracing

was shown, whereas to-day very slight but quite unequivocal waves were registered on the descending line of the tracing. The number of these waves varied according to the length of the so-called diastole or "asphyxial pause," sometimes only two waves appearing, at others as many as six being recorded. That these waves were not the result of either the faulty adjustment of the instrument or tremor of the patient's hand seems evident from the fact that on auscultation over the heart, feeble but distinct cardiac sounds could be heard synchronously with the appearance of the waves on the tracing; and, further, these small waves were too well defined and occurred at such regular intervals as to preclude the possibility of their being due to any extraneous influence. On counting the powerful and feeble cardiac beats, and comparing them with the number of waves on the tracing, the totals were found to agree; the feeble beats were not appreciable to the finger.

October 2nd.—Digitalis in five-drop doses with brandy was given every two hours, but in the course of the day vomiting occurred, and became somewhat urgent; the digitalis was consequently stopped. Vomiting, however, continued, and oral alimentation was suspended, and rectal feeding resorted to.

October 3rd.—For the past two days observations have been made with a view to ascertaining whether the periods of asphyxia were associated in any way with Cheyne-Stokes breathing. But although it was frequently observed that during the period when no pulse was appreciable at the radial, the breathing was very shallow, and that at the onset of the convulsions the respiration became very noisy, nothing like genuine Cheyne-Stokes breathing was observed. On auscultation over the cardiac area to-day, it was observed that during the powerful contractions the sounds were quite audible over the left ventricle, or at least over the point of maximum cardiac impulse; and that over the pulmonic area the second sound was markedly reduplicated, whereas during the feeble contractions the sounds were scarcely, if at all, audible over the left ventricle; but on approaching the pulmonic area they again became distinctly, although very distantly, audible, and here also no reduplication of the second sound could be distinguished.

On carrying the stethoscope up from the pulmonic area to the region of the second right costal cartilage, only one feeble sound was heard; and so far as could be determined by means of a differential stethoscope, this was the first sound. To-day all vomiting has ceased, and oral alimentation has been resumed.

October 4th.—The pulse to-day has become quite regular and of excellent tension and volume. The patient expresses himself as feeling much better, and he sleeps at frequent intervals. The pulse-rate to-day is 72.

October 5th.—Some irregularities were again noted to-day, and the "pulsus bigeminus" has been recorded. The pulse rate is 64.

October 6th.—The tracing taken to-day shows the "pulsus bigeminus" to be well marked. Some irregularities similar to those noted on October 5th were recorded.

October 9th.—Pulse is quite regular, numbering 72 per minute. On auscultation over the heart, the first sound is heard to have a murmurish termination, and the second sound is reduplicated over the pulmonic area. The apex impulse is visible and tangible in the fifth inter-space, three quarters of an inch to the inner side of the nipple line. Respirations number 20 per minute. There is nothing in the condition of the lungs calling for note. The urine contains no albumen and no sugar.

October 10th.—The patient was dismissed from hospital to-day, and consequently regular observation of the case was discontinued, but the patient was seen from time to time, when the pulse was perfectly regular, and for several weeks he remained fairly well, having been warned to avoid over-exertion.

December 3rd.—The patient was seen to-day, when he complained of the old symptoms having returned some three days ago. He looks ill, his face is pallid, the lips are cyanosed, the eyes sunken, and the tongue is coated with a whitish fur, constipation has been the rule of late, the temperature is normal. He has had a slight cough, but this has been unaccompanied by any expectoration. He had had some threatenings of convulsive seizures, that is, he had felt faint, his face had become pale, but he had not lost consciousness.

These attacks had been of very frequent occurrence. The pulse is irregular, as appreciable to the finger at the wrist, and very slow, but the intervals between the beats do not as a rule last longer than ten seconds. The rate of the pulse varies from 15 to 24 per minute. As a rule there are, however, periods during which the pulse numbers 44 per minute at the wrist. Simultaneous cardiac auscultation detected quite distinct though very feeble cardiac contractions, equal in number to the powerful systoles. Auscultation over the apex impulse, which was situated in the fifth space, half an inch to the outer side of the vertical nipple line, during the powerful cardiac contractions, both sounds were muffled and leathery in quality, but no definite murmur was audible. Over the pulmonic area the second sound was doubled, and accentuated as to its second element. Over the aortic area the second sound was much less pronounced. During the feeble cardiac contractions the point over which the heart's sounds were heard with greatest intensity was at mid-sternum, on a level with the fourth inter-space and fifth rib. The sounds were also heard, but less distinctly, over a triangular area which had for its boundaries three lines joining the following points: The fourth sterno-costal articulation on the left side, the fifth sterno-costal articulation on the right side, and a point at mid-sternum on a level with the sixth rib. Over the whole of this area both heart's sounds (during the feeble systoles) were clearly audible, the second element being the more distinct at the upper angle of the area. On carrying the stethoscope upwards towards the second right costal cartilage the sounds were still heard, but much less distinctly. Also, on listening over the fifth inter-space (where the cardiac impulse was visible during the powerful systoles), there was much diminution of intensity of the feeble sounds.

January 5th.—To-day the patient complained of pain in the epigastrium, breathlessness on exertion, and cough, which was accompanied by a clear expectoration. On examining the abdomen the dulness in the hepatic region was found to measure $5\frac{1}{2}$ inches in the middle line and $6\frac{3}{4}$ inches in mammillary line. Pressure over this area elicited an expression of pain. There was some slight impairment of percussion sound

over the extreme base of the right lung behind. Some fine rales were audible over both bases posteriorly. The urine was turbid, and contained a slight amount of albumen.

January 18th.—There is evident improvement so far as the rhythm of the powerful beat is concerned, no very long periods elapsing between the powerful beats. For the most part the rate has been about 33, the variations being from 26 to 36 per minute. The impairment of the breath sounds and diminished percussion resonance at the right base is rather more marked. The hepatic dulness now measures 7 inches in the nipple line and 5½ at mid-sternum. On auscultation over the precordium, in addition to the 36 powerful cardiac contractions there were a similar number of feeble systoles; both powerful and feeble systoles were quite regular in point of time. The pulse rate was 36.

February 1st.—The patient took exercise to-day, but on returning home he was rather exhausted; the pulse was found to be extremely irregular, at times 5 or 6 seconds elapsing between the beats. During these intervals feeble cardiac systoles were audible, varying in number from 2 to 6.

February 7th.—The pulse is still irregular, but shows a tendency to improvement. There is still very marked pallor of the face and cyanosis of the lips. The measurements of the area of hepatic dulness remain as at last note. The dulness on percussion and enfeeblement of breath sounds over the base of the right lung behind remains much as previously noted.

February 10th.—To day the pulse is noted to be of a very variable rate, sometimes numbering 18 per minute, at others perfectly regular at 33. Cardiac auscultation, however, reveals the fact that the heart beats number from 90 to 100 per minute, showing an average of 3 or 4 feeble beats to each powerful systole. There is some increase in the extent of the dulness of the right lung behind. An exploratory puncture was made over the area of dulness at the extreme base, but no fluid was obtained, the needle apparently penetrating dense tissue. In addition to the facts already described as existing in the cardiac condition, there is observed to-day a murmur, soft and blowing in quality and systolic in rhythm, heard generally over the precordium; over the lower end of the

sternum the murmur has a tendency to a coarser and rougher quality.

March 26th.—The pulse to-day numbers 48 per minute, the cardiac contractions numbering 96. The systolic murmur observed on February 10th is not so marked. Percussion dulness of the right lung behind now extends as high as the lower angle of the scapula. On auscultation over this region the main features noted are deficiency of breath sounds with distant tubularity. Vocal resonance and fremitus are decreased, and there is lessened movement of the right side of the chest during inspiration.

July 1st, 1895.—The pulse rate is 33 per minute, with one or two feeble systoles interposed between each powerful cardiac contraction; shortness of breath on exertion has been urgent. The hepatic dulness still measures 7½ inches in the vertical nipple line, the condition at the base of the right lung indicates improvement, there is now only slight impairment of percussion resonance, and breath sounds are merely deficient. The cardiac murmur has now disappeared, and the facial pallor is replaced by a fairly healthy colour. He has had a tendency to faintness on only two occasions recently.

October 1st, 1895.—The pulse numbers 33 and is quite regular. The cardiac contractions number 66, indicating one feeble systole to each powerful contraction. Beyond this fact the cardiac condition presents nothing calling for note. There have been no seizures since last note, nor any tendency to faintness. Dyspnoea has been present for the most part. The area of hepatic dulness is the same as at last note, and the condition at the base of the right lung remains as before. The urine is free from albumen. The patient is now able to do some light work. He is now able, with some reliance, to count his pulse, that is so far as the powerful beats are concerned, and he states that it has never been lower than 33 to the minute for the last three months. He still complains of a sense of tightness in the epigastric and right hypochondriac regions; his appetite is good, and there has been no tendency to vomit.

The subsequent history of the case is not known, as the patient died in the course of a few weeks and no autopsy was obtained.

CASE II. Recorded by Stokes.

Synopsis:—Repeated apoplectic attacks, absence of paralysis, remarkable slowness of pulse. Fatty degeneration of both ventricles.

The patient was 68 years of age and had been for a long time the subject of breathlessness. Stupor and a tendency to sleep were characteristics of the condition which had followed an apoplectic attack about three days previously. Breathing was irregular and the pulse numbered 30 per minute. During the seven years previously he had had twenty so-called apoplectic attacks. Before these attacks he was heavy and lethargic, with loss of memory. When they attacked him his pulse would become even slower than usual. He recovered from these attacks without any paralysis. Oedema of the feet developed and dyspnoea became urgent. Later he was suddenly seized with an attack and died in the course of a few hours. The autopsy discovered the substance of the brain to be of a yellowish colour and somewhat watery. The lateral ventricles contained some fluid, but did not seem to be dilated, although the communicating foramen was enlarged. The lungs presented nothing of importance. Some fluid was found in the anterior mediastinum. The coats of the carotid and middle arteries of the dura were white and opaque from deposition of calcareous material. The right auricle was much dilated. The wall of the right ventricle was the seat of marked fatty degeneration. The left ventricle was also much degenerated and very thin. The aorta was the seat of calcareous deposit. No valvular defect was observed.

CASE III. By Stokes.

Synopsis:—Repeated pseudo-apoplectic attacks, not followed by paralysis; slow pulse with valvular murmur.

The patient was a man 60 years of age, who had enjoyed good health until three years previously, when he was suddenly seized with a fainting fit, which would have caused him to fall had he not been supported. These attacks occurred several times in the day and seemed to leave no ill effects. Since that time

he had never been free from such attacks for any length of time, and had experienced at least fifty similar seizures. They are uncertain as to their period of invasion and very irregular as to their intensity, some being very much milder and of shorter duration than others. He was never convulsed and there was never any frothing at the mouth. The duration of the attack is seldom more than four or five minutes, sometimes less. During that time he is quite unconscious.

Cardiac condition.—The impulse is slow, and of a dull and prolonged heaving action. First sound is accompanied by a soft murmur. The second sound is also slightly imperfect. The pulse rate is 28 per minute, prolonged and sluggish in character. The urine is free from albumen, with a specific gravity 1010. Later: Pulse varied from 28 to 30 per minute. There have been two threatenings of fits since admission, both occurring in bed, and which have been warded off by a peculiar manoeuvre. As soon as the patient perceives symptoms of the approaching attack he turns on to his hands and knees, and by keeping his head low he says he often averts what otherwise would end in an attack. In addition to the regular cardiac contractions occasional semi-beats are observed, very feeble, and unattended by impulse, and corresponding to a similar state of the pulse, which thus probably amounts to 36, the evident beats numbering 28. Later: Occasional abortive attempts at contraction were observed, probably amounting to about four per minute. They do not destroy the regular intervals between the stronger sounds, but are heard as it were filling up the spaces. A corresponding state of the pulse could not be recognised.

The patient again came under observation three months later, when the following facts were noted: There was a remarkable pulsation in the jugular vein. The number of reflex pulsations was difficult to establish, but it was more than double the number of the ventricular contractions. About every third pulsation was strong and sudden, the other waves being less distinct. These probably corresponded with the contractions already noticed in the heart. The patient had had scarcely any cardiac attacks in the intervals, but he had experienced premonitory sensations.

CASE IV. By Burnett.

Case of Epilepsy with remarkable slowness of pulse.

The patient was a naval officer, aged 46, who had had a single attack of epilepsy 16 years previously. Had a second attack in August 1820. In January 1821 he had five paroxysms in the course of half an hour. These attacks were epileptiform in character. Four months later the paroxysms were slighter and less frequent. He experienced a sense of uneasiness in the epigastrium and dyspnoea. The pulse numbered 36 per minute. On the following day the pulse numbered 20 as a rule, and ranged to 32. During the following week the pulse rate was varied between 28 and 56. There were no seizures during this time.

July 2nd. (Two months later).—Four attacks occurred. The pulse then numbered 56.

July 3rd.—Pulse 24, face very shallow, great tenderness on pressure over hepatic region.

July 9th.—Several attacks, pulse 52.

July 16th.—Frequent slight attacks, epigastric uneasiness continues.

July 17th.—Pulse rate 18.

July 20th.—Several severe paroxysms; pulse rate 14.

July 22nd.—Paroxysms of more frequent occurrence and of greater severity. During the paroxysms the pulse is altogether suspended, face becomes pale and convulsed, and a transient flush then succeeds. The pulse is again felt, and patient regains consciousness, to be again attacked; the pulse beats 74 a minute for perhaps the space of a minute, then it intermits for 7, 8, or 10 seconds. Later in the day the attacks continued and the pulse rate was 20 per minute.

July 24th.—The pulse varies from 16 to 18. Attacks are frequent.

July 25th.—Attacks are frequent, but more like spasmodic twitchings than epilepsy. The pulse is often suspended for 10 or 12 seconds.

August 2nd.—Twitchings continue, but no severe attacks have occurred. Pulse rate 24 a minute. The subsequent

history of the case is unknown beyond the fact that he developed anasareous swellings.

Burnett makes reference to a case reported by Morgagni, in which a condition of slow pulse with epileptic attacks is described in a priest of 66 years of age, another feature being pain in the right hypochondriac region. Morgagni does not mention the rate of the pulse, but Burnett infers from a quotation from Gerbezius that it was not below 24 per minute.

CASE V. By Holberton.

The patient was a gentleman, aged 64, who was thrown from his horse and sustained an injury to his neck in December 1834. He experienced pain and stiffness in the neck, he could not rotate the head, and was unable to move in bed. He recovered from the effect of the injury to a great extent; difficulty in moving the head continued for a considerable time. Beyond this nothing of note occurred in the progress of the case for some two years.

In January, whilst out walking, he had a fainting fit. The medical attendant found the pulse beating at the rate of about 20 per minute. A few months later he had a second attack. Holberton first saw the patient in March 1837, when the pulse numbered 33 per minute. This rate was readily altered by certain conditions—notably mental excitement increased the rate, which was followed by a slowing and often by a fainting fit. The general character of the pulse when the patient felt well was full, free, sometimes regular, at other times intermittent. Any disorder of the functions readily affected the pulse and determined the tendency to faint. The attacks increased in frequency as time advanced, and in June 1838 an alarming succession of fits occurred and continued at intervals of from 1 to 15 minutes. The pulse rate fell from 15 or 10 in the minute, which was usual, to 12, 10, 9, or 8, and on several occasions when the patient was quite sensible it was as low as 7½. If the finger were placed on the radial artery the approach of a fit might be known sometimes for a second or two before it manifested itself by any change in the countenance. The pulse would cease before the syncope took

place, and the fit would continue until the heart beat again, when the face would redden and consciousness return with a wild stare and occasionally a snorting and a convulsive action of the muscles of the mouth and face.

The frequency of the attacks was uncertain, sometimes the patient would have two or three in a day; at others only one in two or three days; again perhaps only one in a week or in a fortnight or in a month. Sometimes the fit would be severe and all consciousness lost, at other times there would be a mere threatening of giddiness. He had two particularly severe attacks, one six months before death, the other terminating fatally.

Autopsy.—The *lungs* were healthy. The *heart* was large; the left ventricle was rather thin; the aortic valve was healthy; the right auriculo-ventricular opening was very large, and admitted the points of four fingers and thumb. The left auriculo-ventricular opening was enlarged, admitting three fingers easily. The lining membrane of both ventricles was thickened.

The cranium was very thin, the dura mater was firmly adherent throughout, the cavity of the arachnoid contained a large quantity of serum. Brain substance was healthy; the medulla oblongata was small in size and firm in consistence. The foramen magnum was altered in shape. The antero-posterior diameter was so much narrowed that it would not admit the little finger. There was a general thickening about the dura mater and ligament covering the posterior body of the axis. The articular surfaces of the atlas were firmly ossified to the occipital bone, and permitted no movement whatever. There was no calcareous deposit in the vascular system. The pneumogastric nerves were large, and the middle cervical ganglion on the right was unusually developed.

CASE VI. By Tripier.

Epilepsy and slow pulse with cardiac arrhythmia.

The patient was a commercial traveller, 45 years of age. Previous history indicated convulsions during childhood. For

nearly seven years nervous crises occurred at irregular intervals, but frequently accompanied by a slow pulse. The pulse as a rule did not exceed 60 beats per minute, and at the times of the crises fell to 18, 16, 14, and 12 beats per minute; the maximum slowing coincided with the greatest frequency of the crises. These crises frequently followed each other in rapid succession and were occasionally so slight as not to interrupt conversation, although the patient sometimes lost consciousness momentarily. The radial pulse was 17 per minute and feeble. The cardiac impulse was not appreciable to the hand. On auscultation over the precordium in the region of the nipple the cardiac beats were heard synchronously with the pulse, but distant and indistinct. Nearer the sternum the same sounds were still heard, but in addition more frequent beats became audible, to the number of 84 per minute. These sounds resembled the beating of another heart. The second sound was not appreciable; these (weaker) beatings were more marked on nearing the sternum, where they attained their maximum intensity. They were also heard to the right of the sternum. They are more clearly struck than those heard towards the nipple. There were pulsations in the veins in the neck, synchronous with the frequent beats, and also other beats more slow.

The patient died suddenly, and nothing of importance was found at the autopsy; both the brain and the heart appeared normal, although no microscopical examination of the heart muscle was made.

CASE VII. By Tripier.

Epilepsy with slow pulse and coupled rhythm of the heart.

The patient was a woman 53 years of age, and had typhoid fever at 9 years of age and jaundice at 18. Had suffered from dropsy of face and legs at different periods.

Physical condition.—Some oedema of the lower limbs; the heart did not appear to be enlarged. A soft systolic murmur was heard over the precordium, more marked over the second and third left inter-spaces close to the sternum. Lungs

appeared healthy. Urine was scanty, and contained albumen, blood, and renal débris. In a few days after coming under observation she had an attack of faintness with general tonic spasm of the muscles, but unattended by clonic spasm. On the next day pulse numbered 72, of fair tension, but failing; more attacks of syncope and muscular twitchings, which recurred at frequent intervals. Later syncopal crises were accompanied by loss of consciousness and followed rapidly by cyanosis of the face, but no paralysis. The pulse numbered 44 per minute and was regular. Auscultation of the heart detected strong and feeble sounds. Normal systolic and diastolic sounds were followed by weaker and shorter sounds, diastolic and systolic The strong systolic sounds correspond with the pulse, but the feeble sounds are not associated with any appearance of radial pulse. Sphygmograms do not indicate any beat during the feeble systole; the heart sounds have never the character of separate sounds. It is a question of a double cardiac revolution giving rise to a single arterial pulse. Thus 44 radial pulse beats and 88 cardiac beats are counted per minute. The following is the order of the occurrence of the sounds and their relation to the silences:

1st. Strong systolic sound, short silence.
2nd. Strong diastolic sound, short silence.
3rd. Feeble systolic sound, short silence.
4th. Feeble diastolic sound, long interval,

and so on.

The feeble sounds are like the echo of the strong sounds, and audible over the precordium. The systolic murmur was audible only during the powerful contractions. The cardiac impulse was plainly felt during the strong systoles, but doubtfully during the feeble ones. Jugular pulsation was seen during both strong and weak beats. Minor crises were accompanied by passing unconsciousness, fixity of the eyeballs, immobility, and latterly dilation of the pupil.

The progress of the case was attended by gastric disturbance; the patient died with symptoms of general weakness.

Autopsy.—Brain. Meninges slightly thickened and adherent at the base. Middle cerebral and basilar arteries atheromatous;

small haemorrhage of the right side of the pons. *Heart* was enlarged, flabby, and presented marked fatty degeneration. Mitral valve was thickened, the orifice being dilated. The left ventricle was enlarged and the walls thickened. The aortic valves were competent. The aorta was atheromatous; the right ventricle and tricuspid valve were dilated. *Lungs, liver, and kidneys* presented nothing of importance.

In addition to the foregoing cases recorded by Tripier that observer has collected a large number of cases, and from these reports he endeavours to demonstrate the relationship of the epileptiform crises to the cardiac disturbance, and more especially the association of these crises with the double and triple cardiac rhythm. But on Tripier's own showing the majority of these cases have not been reported with sufficient exactitude with regard to the points which they are intended to demonstrate; consequently we have contented ourselves with dealing with Tripier's observations and conclusions concerning these cases, which will be discussed later.

CASE VIII. By St. George Mivart.

Epileptiform seizures with unusually slow pulse.

The patient was 61 years of age, and had served in the army and police force. There was no history of neurosis in the family; he had never had syphilis nor rheumatism. He was of temperate habits and had enjoyed fair health, except that "he suffered from his heart," which he attributed to hard work and exposure. Palpitation had been frequent and usually occurred at night. The first fit occurred six years prior to his coming under observation. The second attack occurred two years later; stooping and lowering of the head encouraged the attacks. The last seizure occurred the day prior to his being seen: whilst sitting on a chair he fell forward and had three attacks in succession. The seizures were preceded by a buzzing in the head and a feeling of being unable to stand, with giddiness.

Physical condition.—He looked anaemic, the pupils were equal, the urine was normal, the temperature was also normal,

the tongue was coated. The pulse was regular, firm, and full between the beats, which numbered 20 per minute. The heart's impulse was almost imperceptible, there being only a faint movement at the tip of the sternum. Auscultation revealed apparently the presence of one sound synchronous with the pulse, and heard best towards the apex. One month later the pulse numbered 24, a systolic bruit was heard over the heart, and apparently an aortic systolic bruit. At this time the urine contained a slight amount of albumen. Two months later the pulse rate was 24. The patient was never observed during a seizure, and no note of the pulse either prior to or during the attack was recorded.

CASE IX. By Gibbings.

Remarkably slow pulse with epileptiform seizure.

The patient was 66 years of age, and had a well marked arcus senilis. There was no family history of neurosis. He had had no specific disease, and neither gout nor rheumatism. Dyspnoea was a feature. The pulse was 60 and intermittent. The urine was normal; the heart's sounds were normal; respiratory sounds also normal. Six months later the pulse was 44 and regular, and the patient had frequent feelings of faintness. Later, dyspnoea became urgent. The pulse rate fell to 34, but was regular and synchronous with the heart's beats. The pulse at a later period became slower, and frequent epileptiform attacks occurred. During these attacks the face became pale, the pupils dilated, and the eyes fixed, and the pulse imperceptible at the wrist. After a few seconds the pulse returned, feebly, the face then flushed all over, and clonic convulsive tremors ensued lasting for 5 or 6 seconds.

The first indication of an attack was the non-recurrence of the usual pulse beat. Almost directly there was a moaning sound and the face became fixed. The sequence was observed frequently, so that with the finger upon the pulse it was possible to know when the attack was beginning a second or two before the face changed. Later, the pulse fell to 22 per minute, but was full and regular, the heart's beats corre-

sponding in time. No cardiac lesion was discovered. The attacks became frequent and distressing, occurring both on exertion and during rest. The pulse rate continued to fall, in fact until only 12 and 13 beats were counted per minute. Later, there was an improvement in this respect, 30 to 34 beats per minute being noted. He died suddenly in a faint.

Autopsy.—The right pleura contained some fluid. With this exception all the organs were apparently normal. Dr. Moxon performed the sectio, which is a sufficient guarantee that no gross lesion was overlooked.

CASE X. By Bristowe.

Slow pulse with epileptiform seizures.

The patient was 31 years of age, and had been a soldier. There was a history of both syphilis and "fever and ague." He had not had rheumatism, nor had he suffered from palpitation or breathlessness until a few days before he was seen, when he had an attack of faintness caused by over-exertion. Later, he had another attack and lost consciousness. The attacks became more frequent, and he was compelled to give up work. Faintness at the chest seems to have been the first symptom of an attack; this was followed by flushing, on the occurrence of which insensibility supervened. The pulse rate as a rule was 26 per minute, and attacks of fits were frequent. These came on at the end of a period during which there was a considerable interval between the successive cardiac beats. While the pulse was at the rate of even one beat in 3 or 4 seconds no fit occurred, but whenever (and only when) the intermission was prolonged to 5 seconds, a fit followed at the end of that period. A loud roaring systolic murmur was audible above and to the right of the apex, and prolonged thence to the base. Later, the fits had been of very frequent occurrence, and it was noted that prior to the onset of the attack the face became pale and then flushed, and whilst insensibility lasted the hands twitched slightly. Occasionally the pulse beat at the rate of 80 per minute, quite regularly for a few beats, then came a period when one or two beats

occurred at the rate of one or two seconds. Later, the attacks had been more frequent, and on listening to the heart its sounds correspond accurately with the pulsations at the wrist, but between all the beats (attended by waves perceptible at the wrist), separated from one another by intervals of between one and four seconds, there are one, two, or three slight impulses of the heart distinctly to be felt, and seen by the imparted movements of the stethoscope. These latter beats are wholly unattended by cardiac sounds or pulsation in the arteries. Apparently the action of the heart is quite regular in its rhythm. These intermediate impulses, felt at the base of the heart, are they due to the action of the auricle or to the hardening of the relaxed but empty ventricle?

CASE XI. By Hodgson.

The patient was 44 years of age, and came under observation supposed to be suffering from cirrhosis of the liver and kidneys. At first the pulse rate was 108 per minute. Later, it fell to 52 and became intermittent, and continued to fall as low as 24 and 28. Pericardial effusion then developed; following this the pulse rose to 36. Two fits were recorded at this time. Six months later he was again seen, suffering from syncope and fits.

When the pulse stopped for 15 seconds the patient became faint, but when the pause lasted 30 seconds epileptiform spasms ensued. The attacks varied in frequency and severity—sometimes many hours elapsed without the occurrence of a crisis; at other times the seizures and syncopes were of almost constant occurrence, frequently only a few seconds elapsing between two attacks. As a rule the pulse rate was about 21 per minute. On one occasion, however, it was noted to have fallen to 6 per minute. Sphygmograms were taken to show the length of the pause and the fall of the blood pressure. Auscultation and tracings show that the heart was in a state of a systole at the time of the pause. The pulse rate never rose above 36. A gumma of the rib was discovered. The subsequent history of the case was not given.

CASE XII. By Jones and Clinch.

The patient was a man, aged 60 years, who had been an inmate of an asylum for some ten years. He appears to have been addicted to alcohol, and he was also the probable subject of syphilis. After a residence in the asylum of ten years he was suddenly taken ill whilst at a meal with a syncopal attack. Slowness of the pulse was noted, the rate varying between 40-26-20-11 beats a minute. Breathing was laborious. This state was followed by a series of epileptiform attacks, and it was noted that the time of onset of the fits was preceded by a considerable interval between the pulse beats. On one occasion fifteen seconds elapsed between two beats. During one minute eight beats were counted on auscultation. During the fit, the expression was of mental anguish, with protrusion of eyes and dilated pupils. Flushing of the malar regions was marked. The respirations were noisy, slow, and laboured. Subsequently to a fit, the pulse rate increased to 50-60 per minute. The fits varied in intensity and extent—sometimes only slight unilateral facial twitching, at others the convulsion was general. Physical examination revealed: The cardiac dulness was increased both to the right and left, and on auscultation an occasional hard presystolic murmur was detected. The second aortic sound was sharp. The pulse was slow, large, collapsing, and of low tension. Degeneration of the arterial coats was very marked. Lungs—right, deficient resonance from the apex to the base, and medium-sized rales on auscultation. Examination of the nervous system detected no evidence of paresis. The right knee jerk was absent, the left was present; ankle clonus was elicited on both sides. The patient lived for two weeks after the onset of the first convulsion, and a period of six days elapsed without the occurrence of an attack. The pulse varied between 21 and 58. A few days later engorgement of the right lung increased. Right ptosis and contractions of the right occipito-frontalis muscle were noted. Laryngo-pharyngeal palsy became evident. Conjugate deviation of the head and eyes to the right, and some jerking of the right

sterno-mastoid; alternate flushing and palsy of the face, and slight obliteration of the right naso-labial fold were all features of the case leading up to a fatal issue. The necropsy revealed softening and anaemia of the brain substance generally. The basal vessels were not markedly diseased. The heart was large, dilated, and the walls had undergone fatty degeneration. The valvular orifices were dilated and the mitral and aortic valves were slightly thickened. The aorta was dilated, the lungs were congested and oedematous, and old, scattered, tubercles were detected. The left kidney was markedly caseous. The spinal cord was small, and the second right sacral ganglion was converted into a cyst, and a small tumour was found in the third sacral root.

CASE XIII.

Pugin Thornton reports the case of a woman, aged 30, who had had tracheotomy performed for syphilis of the upper air passages in 1872. At that time the pulse rate was 40, and six weeks later it had fallen to 16-24 a minute. It appeared that the patient had been under the care of Dr. Ransome of Nottingham, in 1870, and his notes of the case are very interesting and descriptive. The patient had attacks of an epileptiform nature, associated with a very slow rate of the pulse, the average rate being about 24 beats per minute. The attacks lasted for a period of two months. A severe fit began with sudden pallor of the face, complete loss of consciousness and motor power, cessation of the heart's action for several seconds, on one occasion for 18 seconds. Respiration then became quickened and almost stertorous, the face flushed, the eyes suffused, fixed and turned upwards. She foamed at the mouth. After a time consciousness returned, the expression became calm, and no signs of distress remained; the intellect became clear, and the pulse returned to the normal rate of 24 per minute. Hallucinations were frequent. The intensity of the fits varied—sometimes twelve occurred in a quarter of an hour—and were of a transient nature, and the patient could take up the thread of conversation which had been interrupted by the attack.

Wilkinson reports a case of a man, aet. 62, with a pulse rate of 26-38, with suppression of alternate beats, some of the second beats being palpable, and seen on the sphygmogram. The patient occasionally lost consciousness, but had no fits and no spasms; the attacks are said to have lasted five minutes. The patient frequently fell on the back of his head.

The earliest record which we have been able to find of this type of disease is that described by Burnett in 1825. There can be little doubt that this was clearly a case of inhibited cardiac action immediately preceding the occurrence of epileptiform convulsions; the sequence of events being failure of the pulse, as appreciable to the finger at the wrist, pallor of the face, onset of convulsions, and then apparently simultaneously a subsidence of the convulsion with reappearance of the pulse, flushing of the face, and return to consciousness; the intervals which elapsed between the stoppage of the pulse and the onset of the convulsions varying in length from 10 to 15 seconds. In the uncertainty of their occurrence, and in the varying degrees in the severity of the attacks this case closely resembles the one already recorded; another point of similarity being that at times the pulse beat at the rate of 74 per minute and quite regularly, and then suddenly there was a complete suspension for many seconds; a fact of some importance also being that sometimes the attacks were confined to slight spasmodic twitchings.

Holberton's case is equally clearly one of epileptiform crises following the temporary suspension of the pulse. The observation that an impending attack might be predicted by keeping the finger on the pulse when the latter was lost for a second or two prior to the commencement of the convulsion—the pallor of the face being succeeded by the flushing and the simultaneous reappearance of the pulse, and the return to consciousness with a wild stare—is almost identical with the facts as observed in Case I. Another point of resemblance was the uncertainty of the time of occurrence and the variability in the degree of severity of the crises.

Of the two cases reported by Stokes, although both appear to be cases similar to those of Holberton and Burnett, neither is

reported with sufficient exactitude to allow of any definite opinion being formed; notably no information is afforded as to the state of the pulse immediately prior to the occurrence of the seizures. In one of these cases the so-called pseudo-apoplectic seizures were probably of the nature of epileptiform spasms accompanied by loss of consciousness, more especially as such seizures were of frequent occurrence and left no paralytic sequelae. The other case is interesting, for here there is a condition in which a pulse of 28 to 30 per minute obtains (with more or less feeble cardiac contractions interposed), and in which during the attacks consciousness was frequently lost, but at no time was convulsive spasm observed. In this particular the case was characterised by the occurrence of the minor degree of the critical manifestations, many of which were observed by Holberton, Burnett, and the writer. The manner in which the patient was frequently able to ward off impending seizures by elevating the trunk and lowering the head points very strongly in the direction of the insufficient supply of arterial blood to the brain as being the cause of the attacks. The statement that the attacks never lasted longer than 5 minutes is ambiguous, and probably during such a lengthened period the patient was rapidly passing from one syncopal attack into another.

St. George Mivart's case is apparently one in which a slow rate of pulse existed, with or without feeble cardiac systoles being interposed, and was associated with faintness and epileptiform manifestations. The fact that this patient was never seen during an attack by the recorder of the case deprives this report of much of its value, as we have no information as to whether there was any lengthened suspension of the pulse prior to the attacks. The observation that the pulse which beat at the rate of 20 per minute was full and firm between the beats is interesting, and is quite contrary to the facts noted in the present case. Gibbing's case so closely resembles those of Holberton and Burnett that it is scarcely necessary to do more than indicate this fact. One point, however, in this record is the fact that the patient generally experienced an aura which consisted in the sensation as if hot fluid were trickling down the neck.

Considerable interest and importance attach to the report of Bristowe's case, more especially as that observer lays great stress on the fact that, while the pulse was beating at the rate of 1 beat in 3 or 4 seconds, no fit occurred ; but that whenever, and only when, the intermission was prolonged to 5 seconds an epileptiform seizure followed at the end of that period. It would appear, however, that with such short periods of suspension of the pulse many of these attacks were of the nature of minor epileptiform manifestations.

Hodgson's case is scarcely of less importance and interest, and is in striking contrast to that reported by Bristowe, for here such periods as 15 seconds of pulse suspension were merely associated with faintness, and epileptiform attacks only supervened after such lengthened periods of pulselessness as 30 seconds. This case, in some ways perhaps, more closely resembles our own than do any of the others. Notably it presented these exceptionally long periods of asphyxia ; another point of similarity being that on occasions the seizures occurred in such rapid succession that only a second or two elapsed between two attacks. In this case also we have the first mention of the fall in the blood pressure being indicated on the sphygmographic tracing.

Jones and Clinch's case is interesting in that it is accompanied by sphygmographic evidence, which is conspicuous by its absence from most of the other records. Here we note also that considerable intervals, sometimes as long as 15 seconds, elapsed between pulse beats, and the lowest rate of cardiac contraction was eight beats per minute (by auscultation). On some occasions, however, the pulse rate rose to 60 per minute. The variation in intensity and extent of the convulsive attacks is very notable.

Pugin Thornton and Ransome's case is remarkable for the similarity of the description of the convulsive attacks to those of Holberton and Burnett.

The case reported by Day was another of those in which very long periods of asphyxia preceded the convulsive attacks and the crescendo reappearance of the pulse on the return to consciousness.

Before referring to Tripier's cases it will be here convenient

to give a brief description of the tracings accompanying this paper, as it will be necessary to refer to them in discussing Tripier's observations.

Tracing No. 1 (*September 23rd*, 1894) was taken soon after the patient first came under observation, and indicates the termination of a period during which the pulse was quite regular, three of such beats being registered. Then follows a period during which the pulse is apparently absolutely in abeyance. These periods of asphyxia were of variable duration—sometimes 10 or 12 seconds, at others only 4 or 5 seconds elapsing between the pulse beats, this state of matters lasting for 30 or 40 seconds; then a number of more powerful beats are registered, increasing in force in a crescendo manner, to be again succeeded by a series of beats at intervals of 4 or 5 seconds. During the greater part of the time when this tracing was being taken the patient lay quite still. Facial pallor was very marked, and although consciousness was not lost and no convulsive spasm was observed, a certain degree of mental inactivity or stupor was evident.

Tracing No. 2 indicates three long periods of asphyxia, separated by short intervals, during which more or less powerful beats are recorded. The return of the pulse in a crescendo manner is well demonstrated. During these long pauses the facial pallor gradually increased until it became almost death-like. Unconsciousness gradually supervened, and towards the end of the pause epileptiform spasm ensued, commencing in the muscles of the face, but being confined to the head and neck; the return to consciousness being heralded by the return of the pulse and the coincident flushing of the face.

Tracing No. 3 presents a combination of the characters exhibited by the two preceding ones, the periods of asphyxia varying in length in a most marked manner.

Tracing No. 4 shows a pulse of excellent tension and of perfect regularity.

Tracing No. 5 indicates also a pulse of good tension, but here and there it exhibits a tendency to the character of the "pulsus bigeminus."

Tracing No. 6 demonstrates the bigeminal character in a more perfect manner.

Tracing No. 7 indicates a reversion to the type seen in Nos. 1, 2, and 3.

Tracing No. 8 shows a general pulse rate of approximately 20 per minute, but with numerous small waves recorded on the down stroke, or diastolic part of the tracing. These waves vary greatly in number, and correspond in point of time with the occurrence of feeble cardiac sounds, which were audible on auscultation over the precordium.

Tracing No. 9 also demonstrates the small waves on the descending line.

Tracing No. 10, practically similar to No. 9.

Tracing No. 11 in its first part indicates the frequent occurrence of long pauses. Following the third beat there is a gradual fall of the down stroke (indicating a fall of blood pressure). In the first part of the pause, and notably in that part which is above the level of the commencement of the previous percussion stroke, we find small waves registered; these, however, disappear when the blood pressure falls to a certain level. The second half of Tracing No. 11 belongs to an earlier date, September 27th (No. 7), and is included, as it shows the crescendo return of the pulse, with the bigeminal arrangement of the beats after a period of asphyxia.

Tracings Nos. 12, 13, and 14 show pulse beating for a time quite regularly (see No. 14), with a sudden stop and fall of the blood pressure, the pause in one case (No. 14) lasting for nearly 30 seconds, and the record of succeeding beats being distorted by the convulsive action, together with the startled movement of the patient, on the return to consciousness. Convulsions ensued at the latter end of each of the long pauses registered in these tracings, which were the only ones we were able to obtain showing the very long periods of asphyxia and consequent convulsions, as in most cases when the convulsion was very severe the tracings were destroyed by the instrument being displaced from the artery by the spasmodic movements. These tracings were not taken with the view of demonstrating the small waves, but rather to show the length of the pause and the fall of blood pressure.

Tracings Nos. 15, 16, 17, 18, and 19 show the return to regularity and the improvement in tension which occurred

and was maintained until the patient was dismissed from hospital. The modification at the end of tracings Nos. 15 and 18 was adopted to demonstrate the tension by exaggerating the tidal and dicrotic waves.

The remainder of the sphygmograms serve to indicate the varying character of the pulse, both in volume and in rhythm, over a period of many months. Some of them also demonstrate points of importance, which will be referred to in due course.

Although the two cases which Tripier observed himself were reported with the view of supporting the theory that the epileptiform seizures preceded and were the cause of the cardiac arhythmia, it has been necessary to include them in this paper in order that we might examine Tripier's reasons for formulating his opinions. In both of these cases we have a condition in which a slow pulse obtained, and this was associated with epileptiform spasms.

Of the other cases which have been collected by Tripier we do not propose to deal in detail. An extract taken from his remarks on these cases will be sufficient to indicate his views. Concerning one case he says:—"On ne saurait douter que le malade ait eu des crises épileptiformes coincidant avec des troubles cardiaques et notamment avec l'arrêt du coeur et la production d'un rhythme couplé. Ce dernier phénomène est signalé dans l'observation avant les crises, de telle sorte qu'on pourrait croire que celles-ci étaient survenues consécutivement et même qu'elles dépendaient du trouble cardiaque, surtout d'après la manière dont l'observation est redigée. Mais nous rejetons cette interprétation en nous basant sur toutes les observations analogues rapportés précédemment, dans lesquelles nous voyons les crises épileptiques ou syncopales précéder évidemment les déviations du rhythme cardiaque, tandis qu'aucune observation antérieure précise ne permet d'admettre le contraire."

And again:—"Du moment où l'attention n'était pas particulièrement appelée sur les crises, celles-ci ont pu passer inaperçues au début."

And lastly:—"Il n'est guère admissable que les phénomènes convulsifs à répétition, avec perte de connaissance, sans souffrance et sans le souvenir des accidents éprouvés, puissent être

attribués à une lésion du coeur, à une endocardite, fût-elle très évidente, ce qui n'était pas le cas, puisque les valvules se présentaient dans les conditions où on les observe ordinairement avec une hypertrophie aussi considerable du coeur.

"Au contraire, tout s'explique parfaitement avec les crises épileptiformes, à caractères variables et atténués, plus ou moins fréquentes, subintrantes à certains moments, comme on les observe du reste avec les déviations du rhythme cardiaque."

Among the records of the cases which form the basis of these observations by Tripier we find that now famous and frequently quoted one of Holberton. Tripier, while he admits that the facts as recorded by Holberton point to the cardiac failure as preceding the onset of the convulsions, denies the validity of the argument that the cardiac failure was the cause of the following convulsions.

It would appear that nothing could be more explicit than Holberton's description of the pulse failure immediately preceding the convulsion and the subsidence of the latter on the reappearance of the pulse, and we therefore cannot accept Tripier's interpretation of the facts. It certainly would appear unwarrantable to assume that slight epileptiform spasms may have occurred before the pulse failed, and that these minor manifestations were overlooked. As has been previously pointed out, Tripier, in his own records, gives no indication that the pulse was regular before the spasm occurred. Unfortunately, Holberton's case is unaccompanied by any sphygmograms, but in the collection which accompanies this article there are two tracings, one of which, we think, may explain Tripier's views, without in any way invalidating the opposite theory, and the second unquestionably disproves Tripier's statements. In the first of these tracings, numbered 2 in the collection, we have demonstrated three periods of asphyxia, or pauses in the cardiac action (here we make no allusion to any interposed feeble beats, as none are recorded in the tracing, and we propose to revert to this point later); each of these pauses is followed by a series of beats to the number of 7 or 8. The convulsion supervened during the latter part of the pause and was terminating during the occurrence of the earlier beats of this series of 7 or 8; consequently, if the patient were observed, and the pulse

were palpated at this time, only some 3 or 4 beats would be appreciable prior to the cessation of the pulse and the onset of the next syncopal pause. And it would not be an unnatural inference that the convulsion stood to the cardiac failure as cause to effect. But the fact that the convulsion supervened during the period in which the heart's action was in abeyance almost conclusively proves that the cardiac failure was the cause of the convulsion.

If we now refer to Tracing No. 14 we find that at the commencement of this tracing a series of twelve regular beats are recorded; this is followed by the suspension of the pulse for a period of twenty or twenty-five seconds, and during the later part of the pause (that is after a period of some fifteen seconds of asphyxia) the convulsion supervened, to terminate on the recurrence of the pulse. If Tripier's view be correct, a convulsion must have occurred to cause this cardiac stoppage. Such a long pause in the cardiac action as is here indicated would surely have necessitated the occurrence of a very violent convulsive spasm, and it is highly improbable that such a convulsion, or indeed a minor seizure, would have occurred without being observed, or without interfering with the manipulation of the sphygmograph, more especially as we had the advantage of knowing Tripier's views on the subject, and we were naturally careful that even very slight epileptiform manifestations should not escape observation at such a juncture.

It has been pointed out that the convulsion did not terminate immediately the pulse reappeared at the wrist, the explanation of this being that after such a lengthened period of asphyxia as twenty seconds the brain was in such a profound state of anaemia that several cardiac contractions were necessary to re-establish the cerebral arterial circulation to such a degree as to allow of the brain again resuming its functions.

Further, the return to consciousness and subsidence of the convulsion were invariably heralded by this return of the pulse and the flushing replacing the facial pallor.

With regard to Tripier's statement in one of his reports that the greatest frequency of occurrence of the convulsive seizures was coincident with the maximum slowing of the pulse, the following facts may be noted: Tripier makes no reference to

this slowing being accompanied by irregularity. The lowest rate of pulse recorded by him is 12 beats per minute, and still convulsive seizures occurred. We must assume that in Tripier's case the pulse was regular, since he does not furnish us with any evidence to the contrary.

We have now reached one of the most important points in the whole question of the relationship of cardiac derangement to convulsions. The foregoing records have almost conclusively proved that when cerebral anaemia is the cause of the convulsion, the cardiac disability has preceded both. Tripier's views differ entirely from those of the majority of observers with regard to cause and effect, but he is not alone in the view that the greatest frequency of the convulsions coincides with the maximum slowing of the pulse. Broadbent believes that the convulsion is the result of the cerebral anaemia dependent upon the cardiac slowing, as the following quotation abundantly shows:

"The epileptiform attacks are not often violent, but resemble petit mal rather than a typical epileptic fit; while, however, the convulsion may not be so severe, there is profound unconsciousness, not like epileptic coma, but of a syncopal character, and the pulse may be extremely infrequent, sometimes less than 20 in the minute. In my judgment the heart failure as manifested by the slow pulse, and the consequent arrest of the cerebral circulation, are the cause of the fits, and it is not the epileptiform attack that affects the action of the heart."

We see here, then, that while Broadbent differs from Tripier very decidedly with regard to cause and effect, both observers lay much stress upon cardiac slowing associated with convulsions. Balfour, who has had a most extensive experience of bradycardia, tells us that Hope believed that "when one or two beats are regularly and permanently imperceptible in the pulse, such cases contribute the bulk of these cases in which the pulse is described by auscultators as being singularly slow, as, for instance, 30 or 40 per minute." In a few rare cases, however, it is really slow. Balfour's experience would point in the opposite direction. He says: "So far as my experience goes, the rarity has been all the other way, as

I have seen many more really slow hearts than hearts beating at the normal rate with an abnormally slow pulse, due to alternate hemi-systoles." There can be little doubt, therefore, that these two varieties of so-called bradycardia, real and apparent, do exist.

Describing the true form of bradycardia, Balfour says: "As a rule the steady, slow, funereal beat never varies from the time of its commencement until the patient's death." Of the other form he describes the case of an elderly lady whose radial pulse numbered only 20 beats per minute, but whose cardiac contractions were 60 per minute. This lady was the subject of frequent epileptiform seizures. Referring to Holberton's case, he prefaces some remarks on the syncopal attacks by the statement that, although several of his patients had died during a syncopal attack, he had never seen such a seizure. From this we gather that he has never seen the cessation of the pulse for some seconds prior to the onset of convulsion, as described by Holberton. Balfour here raises the most vital point in the whole question. He clearly points out that regularity of the heart's action is a cardinal character of bradycardia, and yet he has never seen syncopal, and probably epileptiform, crises associated with true bradycardia. Dr. Hewan, who was an accurate observer, had a slow pulse habitually, the rate varying from 24 to 32 beats per minute. Yet he never experienced syncopal attacks or convulsions of any kind, and he was able to indulge in such severe physical exercise as mountain climbing. Burnett is very explicit in his statement that sometimes the pulse rate was over 70 per minute, and yet the patient became convulsed. Holberton found the pulse rate as low as eight beats per minute, and yet perfect consciousness was maintained, and presumably he means also that no convulsive seizure occurred. It is interesting to note the frequency with which slow pulse, or bradycardia, or infrequent pulse are described in association with epilepsy in the literature of the past 20 years, but it does not appear that cardiac arrhythmia has been considered a causal factor in the production of the convulsion. Tripier has pointed out that cardiac arrhythmia frequently attends convulsive seizures. He, however, considers that the convulsive attack

precedes the arrhythmia. The problem which presents itself is, how does the cerebral anaemia occur, and how does it give rise to convulsions when a slow pulse obtains? Holberton has recorded a pulse rate of eight per minute, without the occurrence of convulsive seizure. Burnett has recorded a pulse rate of over 70, which was associated with convulsion.

If we refer to the tracing No. 50 of the series which accompanies this article, we see that a hitherto perfectly regular pulse, regular both with regard to its force and rhythm, is followed by a syncopal attack and epileptiform seizure. The epileptiform seizure was certainly of a very modified type, as the asphyxial pause was of short duration. Again, the tracing No. 14 presents a perfectly regular character of pulse in its early part, a regularity which had obtained for a considerable period (at least for several minutes) prior to the onset of convulsions. The pulse rate here was something like 30 per minute. We have frequently seen a pulse rate of less than 20 per minute, and this condition obtaining for several hours or days without the occurrence of the convulsive attacks. We are therefore compelled to find some explanation of the cerebral anaemia which produces the convulsion other than that of simple cardiac slowing. A pulse rate of eight per minute is surely sufficiently slow to induce cerebral anaemia and convulsions. Yet Holberton did not note convulsion when such a condition obtained, but he did note, and he recorded the fact, that for several seconds before the onset of the convulsion the pulse was entirely in abeyance. It would therefore appear more probable that it is the cessation or irregularity of the cardiac action for a period which induces the cerebral anaemia, such cessation, to all practical purposes, as is most perfectly indicated in the earlier sphygmograms accompanying the paper. This is a very different state of matters from the slowness of the pulse, or bradycardia, which Balfour assures us is always associated with regularity.

Our own experience, which extends over many hundreds of observations of the case under discussion, would tend to show that a slow rate of pulse of 18 to 24, *e.g.* Nos. 54, 55, 56, where even a moderate degree of irregularity obtains, is not associated with convulsions. That a slow rate of pulse is

often associated with convulsion is fully established, but it is only where such slowing of the cardiac action is associated with irregularity that epileptiform seizures occur. Irregularity may be either in the way of rhythm or of volume of the pulse, but in these cases it is the irregularity in the force of the cardiac contractions, or in the volume of the pulse, rather than in the rhythm, otherwise it would be difficult to understand exactly when the cerebral anaemia occurs. Take for example a pulse rate of 20 per minute, we have not more than a period of three seconds of asphyxia, and three seconds of asphyxia is not sufficient to induce cerebral anaemia in so marked a degree as to cause convulsive spasm. Bristowe noted this in his case. We would even go further and say that with a pulse rate of even 12 per minute, in which we have a period of five seconds of a pause between each pulse beat, provided perfect regularity obtains, convulsions do not supervene. We would imagine that a pulse rate of twelve beats per minute merits the distinction of slow pulse.

In the early stages of the case under consideration, a period of from five to seven seconds of asphyxia induced convulsive seizure, whereas latterly such a length of pause was only associated with, at the most, cerebral inactivity, or stupor, and a period of 10 to 15 seconds was necessary to induce convulsions. And, of course, the more profound the anaemia, or, in other words, the longer the asphyxial pause, the more general did the convulsion become.

So far there has been a consensus of opinion with regard to the cardiac disturbance preceding and causing the convulsion, Tripier being the only observer who believes otherwise.

We have now come to a point on which there is considerable difference of opinion, viz., the question as to whether the cardiac arrhythmia or slow pulse is associated with abortive cardiac systoles, or interposed feeble beats.

Stokes, Bristowe, and Tripier in their reports all describe the occurrence of feeble beats between the powerful systoles. In one of Stokes' cases, at least, there was one feeble systole between each powerful cardiac contraction. Bristowe describes a number of feeble beats, varying from 1 to 3 between each powerful systole.

Tripier also found several, but probably not more than three, feeble systoles interposed between the strong contractions. In the present case, the number of feeble systoles varied from time to time within very wide limits; sometimes only one weak beat was heard, at others as many as 20. In tracing No. 9, for instance, these feeble beats varied in number from 2 to 5.

Broadbent's view is rather different. He believes that the feeble systoles do occur, but he also believes that a slow rate of pulse may exist without their occurrence, and he quotes the cases of St. George Mivart and Gibbings in support of this view, and certainly these were cases in which very slow pulses, of 24 and 12 per minute respectively, existed. We can only say that no feeble systoles were observed. There certainly appears to be no reason why a slow pulse with irregularity, without any interposed feeble systoles, should not be the cause of epileptiform crises; because, if these feeble beats are not appreciable at the radial pulse, they certainly cannot materially affect the cerebral circulation: so that so far as the effect on the cerebral circulation is concerned, these beats are practically non-existent; and we have, therefore, to all intents and purposes, a condition identical with that in which an irregular and slow pulse obtains. In tracing No. 60, these feeble beats are fairly well brought out; the original tracing, however, demonstrates their occurrence to greater perfection.

We take it for granted that no exception will be taken to the statement that these small waves on the downstroke of the tracing do exist in fact. We must, therefore, endeavour to explain their origin. It has been suggested that they were caused by the rhythmical contraction of the arterial wall. The objection to this explanation is the fact that on auscultation over the precordium distinct, although very feeble, cardiac sounds were audible.

It is scarcely necessary to refute the statement that they may have been due to rhythmical tremors. We are satisfied that during all the asphyxial pauses these feeble cardiac systoles were taking place, and that in the accompanying tracing these small waves are the radial expression of cardiac contractions.

Two points in connection with these feeble beats, as expressed by the sphygmogram, are very striking. In the first place it will be noted that they occur at absolutely regular intervals, between the powerful beats, or on what appears to be the diastolic part of the tracing. Secondly, they are wholly unlike the abortive beats seen in a sphygmogram, taken from a case either of fatty degeneration of the myocardium or mitral disease, where we have an overburdened or feeble muscle making ineffectual efforts to contract. In neither of these conditions do we see the absolutely rhythmic contraction of even the feeblest beats, such as we have here recorded. They bear no resemblance to the feeble beats of a pulsus bigeminus or a pulsus trigeminus, frequently observed after treatment by digitalis; although they did occur after the exhibition of digitalis they were noted before that drug was administered.

We are now confronted with the reasonable objection that if they were the result of feeble cardiac systoles, why were they not indicated in all tracings, and also why were they not recorded during the whole of the intervals between the powerful systoles? We must here refer to tracing No. 9 (which is almost identical with the tracing No. 60). In it we find these small waves recorded on the downstroke in each interval between the powerful systoles. A careful examination of these tracings will discover the fact that at practically no time does the downstroke fall below the level of the commencement of the preceding upstroke, that is to say below the level of the base line, thereby indicating no great fall of arterial tension; and, therefore, so long as we have such a column of blood in the vessel as is indicated by this tension, so long do we find these beats recorded.

If we now examine tracing No. 11 we see following the third powerful beat a long pause; during the earlier part of this pause, before the downstroke has fallen below the level of the base line, these small waves are recorded, but diminishing in size, and ultimately disappearing after the base line is passed.

This gradual fall of the downstroke necessarily means fall of blood pressure, which is synonymous with emptying of the

vessel, so that at the end of the pause there is practically no blood in the vessel, and consequently the means of communicating these small waves is non-existent. What the mechanism of this profound fall in the blood pressure, or outflow of blood to the periphery, for it could not well have been due to a regurgitation, was, will be discussed later. The second part of the objection that these waves were not indicated in all tracings, as, for example, 1, 2, 3, and 14, may be met by the explanation of the great pressure which it was necessary to exert upon the artery to keep the lever on the recording paper during such alterations of blood pressure. Tracing No. 14 demonstrates our point. The first dozen beats, which are situated at the very top of the paper (which indicates that the tension was at its highest), were sufficiently distorted, and yet the pressure necessary to cause this distortion was barely sufficient to keep the lever on the recording paper at the latter end of the long pause, which indicated the minimum of tension, or the maximum fall of blood pressure. It is not difficult to understand that if this pressure effaced some of the details of the powerful beats, it should also entirely obliterate the smaller waves.

We have endeavoured to make it clear that the cardiac irregularity is the primary factor, and that the epileptiform attacks are caused by the cerebral anaemia, resulting from such suspension of the cardiac action. We have also indicated that we are disinclined to the view that the heart's action was entirely in abeyance during the long pauses such as are indicated in the first three tracings.

Hodgson has given it as his opinion that in his case the heart was in a state of asystole, and he assumes it as proved by the absence of any indication of pulse waves, however small, during the pauses. This may be true, but it is not necessarily evidence that no feeble systoles were taking place, and it is difficult to understand how, with such a fall in the blood pressure as must have occurred in 15 or 30 seconds of pause which Hodgson describes, any pulse wave could be expected to be registered. However, we must keep in view the statement that during this time no feeble sounds were audible over the precordium. We have also pointed out that so far as the

cerebral circulation is affected the presence of these feeble systoles is of no importance, as their existence neither increases nor lessens the cerebral anaemia. With regard to the cardiac condition, however, the presence of these feeble beats is of great importance.

In the present case these feeble systoles were accompanied by sounds which were so difficult of appreciation that they frequently escaped observation, and on one occasion a most experienced and skilful auscultator failed to detect them until the time of their occurrence was indicated by small waves on the sphymogram, and this notwithstanding the fact that they were believed to be taking place.

Bristowe, Stokes, and Tripier all describe these feeble beats. Broadbent has also observed them. Tripier would seem to indicate that they might be due to right-sided contraction alone; and his description in one case is very similar to our own, when one powerful systole was followed by a single feeble one. With regard to the occurrence of these feeble beats, Bristowe, at the end of the report of his case, makes the following statement:—"Apparently the action of the heart is quite regular in its rhythm; these intermediate impulses felt at the base of the heart, are they due to the action of the auricle or to the contraction of the relaxed and empty ventricle?"

In our own observations, when it was first discovered that these feeble sounds had their point of maximum intensity situated in the region of the sternum, at a level of the fourth, fifth, and sixth ribs, we were inclined to the possibility of their being due to a right-sided contraction.

This idea was supported by the fact, that during the feeble systoles the second sound was not heard over the aortic area, from which we inferred that the aortic valves were not closing, whilst both sounds were audible over the pulmonic area. (With regard to the description of these sounds, Tripier suggests that they seemed to originate in another heart. Our own impression would be that they resembled the muffled ticking of a watch, or they might be likened to the sounds of a slowly-beating foetal heart.) This view, however, was not tenable for obvious reasons, the chief one being that we had demonstrated oscillations in the column of blood in the radial artery. It

was more probable, therefore, that the left ventricle was contracting, and that, if the ventricle contained any blood, it was the propulsion of this blood against the aortic valves being only sufficiently powerful to raise the aortic curtains. And this puffing out of the aortic valves communicated a wave to the column of blood in the aorta.

This view gained some support from the fact that, so far as we could determine, the second sound, during these feeble beats, was not audible on auscultation over the aortic area.

Another explanation which suggested itself was, that the ventricle was contracting, but that it did not contain sufficient blood even to raise the aortic curtains, and that the wave of muscular contraction was carried along the ventricular wall to the aorta, where it set up an oscillation in the blood column. It is difficult to decide which of these two theories explains the origin of the waves, because during the earlier part of the pause (that is to say, when the pauses were of no great length, as, for example, in tracing No. 60) there could not have been much blood in the ventricle, and yet here the waves were as distinct as those in the later part of the pause. Whereas, on the other hand, at the latter part of the pause, the ventricle must have contained a considerable quantity of blood, as, at the following powerful systole, a considerable volume of blood was thrown into the aorta, as indicated on the tracing by the height of the percussion wave.

Whichever of the explanations be accepted, we are now confronted with the difficulty of explaining, first, the fall in the blood pressure; secondly, the absence of the small waves from the tracing during the latter part of the pause. It did not appear probable that the left ventricle was sending any blood into the aorta. Even although some blood were thrown into the aorta during the feeble systoles, this would scarcely have been sufficient to cause the onward passage of the blood, which is indicated by this fall of pressure.

There can be no question but that the sphygmogram No. 14 indicated a fall of blood pressure, and this means an emptying of the blood vessel. This emptying must either have been of the nature of an onflow of blood to the periphery or to a regurgitation towards the heart. That it was not the latter

seems almost certain, as we had no auscultatory evidence of the fact. Neither does it seem probable that there was a condition of stasis in the arteries; for in that case we should have been able to define the rounded outline of the artery, and this was not possible. The only explanation, therefore, is that there was an outflow of blood to the periphery, and that so long as a certain column of blood remained in the ascending part of the aorta to receive impressions, either from the aortic valves or otherwise, so long did we get these feeble contractions indicated on the tracing. The powerful systoles of the ventricle gave sufficient impetus to the column of blood in the aorta to carry it to the periphery; this being all the more possible owing to the blood passing constantly into a wider vascular area, and through the capillaries into the veins, where the resistance to its onflow was not greater than the impetus which it had acquired from the force of the cardiac contraction.

As soon as the powerful systole was over, the vessels would tend to return to a state of rest, and would only oscillate slightly while there was sufficient blood in the aorta to receive the impressions from the feeble systoles, so that the outflow of blood to the periphery was of a passive nature at the latter part of the pause. Although the fact that the second pulmonic sound indicated that the right ventricle was contracting, and the pulmonic valves opening and closing, it would not appear that this contraction was sufficient to drive the blood through the lungs into the left ventricle; the left ventricle was, nevertheless, receiving a supply of blood, as was indicated by the following pulse, and probably the powerful contraction of the right ventricle during the last systole was sufficient to propel the blood through the pulmonary circuit, and, the tension here being much greater than in the left heart, the onflow of blood was insured, although the feeble action of the left auricle could not supply any *vis a fronte*.

Before proceeding to the question of causation of the cardiac inhibition, there are certain points in the progress of the writer's case which may not inaptly be referred to.

The presence of a systolic murmur, audible over the point of maximum intensity of cardiac impulse, was evidently the result of the enfeebled condition of the ventricle, with dilation

of the mitral orifice, and consequent regurgitation. Another murmur of a rougher quality and more superficial in character, and most marked over the lower end of the sternum, was probably due to tricuspid reflux. Both these murmurs disappeared in the course of time, the pulse having improved in tension and the heart having recovered some of its tone. The sense of fulness and uneasiness in the epigastric region, in the latter stages, was associated with a considerable increase in the measurements of the area of hepatic dulness, and was probably dependent upon venous stasis resulting from the cardiac embarrassment. This engorged condition of the liver and consequent congestion of the portal radicals will probably explain the gastric irritability and intolerance. It is interesting to note that this symptom, referable to the liver, was a feature of Burnett's case, and also of the one referred to by that observer, as recorded by Morgagni. The altered condition at the base of the right lung behind was at first thought to be due to the presence of fluid in the pleural sac. Exploratory puncture, however, did not confirm that idea; it was more probably of the nature of a congestive induration of the lung with thickening of the pleura. Its ultimate disappearance did not give support to the view that it might be of malignant origin.

The record of this case is unfortunately incomplete, as no autopsy was obtained. However, as the chief aim has been to discuss the case from a clinical point of view, this loss, though great, is not sufficient to deprive the case of much of its value; and although we cannot throw any light upon the subject with regard to the probable causation of the cardiac inhibition, from our own experience, this paper might be less complete if we made no reference to the opinions of the observers of other similar cases.

The first autopsy that we find recorded is that on Holberton's case. This is a most important report, for there can be no doubt as to the relationship of the cardiac failure to the epileptiform crises. They stand, unquestionably, as cause to effect. But the information obtained at the autopsy opened up many possibilities with regard to causation. The history of injury, and the symptoms referred to the base of the skull and neck, naturally attracted Holberton's attention, as here he

expected to find the cause of the cardiac disturbance. And there can be no doubt that the alterations which were found in the structures around the foramen magnum and the medulla oblongata suggested very strongly that the pressure thus exerted by the inflammatory products had interfered with the functions of the higher cardiac centre. Fortunately, the examination was made in an extremely careful manner, all organs being subjected to scrutiny. The condition of the heart also affords us valuable information. The left ventricle was thin, the lining membrane of both ventricles was thickened, both auricula-ventricular orifices were enlarged. The patient was a man of 64 years of age. Although Holberton does not report any change in the myocardium, a man of 64 years of age with thin-walled and dilated ventricles and thickened endocardium was probably the subject of degeneration of the cardiac muscle.

An autopsy was obtained in one of the cases reported by Stokes, in which very marked fatty degeneration of the heart was found. Of Tripier's two cases the heart was found to be the seat of extreme fatty degeneration in one case; in the other nothing was found to explain the symptoms, although microscopical examination of the heart muscle does not appear to have been made.

In Gibbing's case nothing was discovered in any organ to account for the symptoms. Microscopical examination of the heart muscle was here also apparently omitted.

Jones and Clinch's case presented a complicated problem on post-mortem examination, but there appears to have been well-marked evidence of syphilis of the brain, and probably also of the vagus and spinal accessory nerves. The heart also was the seat of well-marked fatty degeneration affecting the myocardium. The cerebral vessels at the base were not markedly diseased.

We have endeavoured to demonstrate the relationship of the cardiac disability to the occurrence of the convulsive seizures, and by means of the sphygmograph this has been not altogether impossible. But a greater difficulty presents itself in the explanation of the cause of the cardiac arrhythmia, or slowing. According to our present knowledge, the weight of opinion

probably points to some form of degeneration of the myocardium as being responsible for the cardiac disability. In many of the records of post-mortem examinations on such cases we find evidence of fatty degeneration of the heart muscle. Holberton's case was one in point; Jones and Clinch's is another, but in both of these cases there were other factors at work which would readily explain the cardiac phenomena. In the former case there was a condition of the tissues around the medulla, pointing to pressure on the higher cardiac centre or cardiac nerves. In the latter the observers were satisfied that the spinal accessory and vagus nerves were the seat of syphilitic deposit.

Waller questions whether the vagus communicates directly with the cardiac muscle, or indirectly by the intermediation of ganglia; and he says that "Upon the known fact that the action of the vagus diminishes the frequency and force of the beat, the opinion has been based that the vagus terminates in the muscles as well as in the ganglia, diminution of the frequency being regarded as the sign of vagus action upon the ganglia; diminution of force as the sign of vagus action upon the muscle."

Gaskell believes that all parts of the heart are endowed with the power of rhythmical contraction, independently of the cardiac ganglion cells, but varying in degree in different parts; and he thinks that the ganglion cells occupy a purely accidental position, as a result of their former situation along the wall of the longitudinal blood vessel, or in the least modified part thereof. Quain believed that the form of cardiac disturbance under discussion was dependent upon fatty change of the myocardium. But he thought that the mode in which fatty changes affected the pulse depended upon the part of the muscle affected, and upon the extent of the change.

If we examine the traeings numbered from 20 to 23 and from 41 to 46, it would be difficult to believe that such sphygmograms were the radial expression of the contraction of a degenerated myocardium, or at least of a myocardium so degenerated as to exhibit such vagaries as are portrayed in the earlier sphygmograms. In the tracings 41 to 46 many of the characters are those of high tension, and indicate a power-

fully acting ventricle; and during the whole series of the tracings we have not a single example of the abortive systole so frequently seen in the degenerated muscle tracing, as for example in Balfour's work on *The Senile Heart*, Figs. 3 and 4. Balfour does not regard myocardial degeneration as a cause of true bradycardia; and we take it he believes the arrhythmia, or false bradycardia, to depend upon disturbance of the nervous mechanism also.

The testimony of Morrison and Jones and Clinch endorses Balfour's view.

If we examine the tracing No. 60 it is difficult to resist the impression that the heart is acting in response to two kinds of impulses—the one powerful, the other feeble.

These impulses or their expression occur at perfectly regular intervals, although sometimes there are three, at others two feeble beats following each powerful contraction. The problem which presents itself is, where do these two kinds of impulses arise, the one causing powerful and the other the feeble contractions, and why should they have different expressions? Are these impulses being regularly transmitted from the higher cardiac centre, some being augmented in the heart, either in the intrinsic cardiac ganglia or in the muscle itself; or are the feeble beats the result of stimuli originating in the heart itself, and are the powerful beats the result of such stimuli augmented by impulses from the higher centre, but, owing to some abnormal condition either in the cardiac centre or in the cardiac nerves, these impulses do not originate or are not transmitted with their wonted regularity? This is a problem which can be only suggested here, as the object of this paper has been to deal, as far as possible, with the subject in its clinical aspect.

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